Tyson (Jas.)

HEART-DISEASE OR KIDNEY-DISEASE?

BY

JAMES TYSON, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA, ETC.

FROM

THE MEDICAL NEWS,

July 8, 1893.

[Reprinted from THE MEDICAL NEWS, July 8, 1893.]

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I HAVE myself so often been mistaken in the first conception formed of the relation between a combination of symptoms for which heart or kidney may be responsible, and have so frequently had occasion to share the difficulty with others, that I have been forced to give the matter some thought; and it seemed to me that so practical and important a subject might also be interesting to the members of this Society.

The difficulties met under these circumstances will, I think, be better appreciated by the brief story of four or five illustrative cases:

CASE I.—O. E., a shop-girl, aged fourteen, was admitted to the Hospital of the University of Pennsylvania, October 31, 1892.

Her mother is said to have had heart-disease, but the family history is otherwise negative. The patient herself had measles in childhood, but, with this exception, reported no illness until that for which she sought admission. This set in apparently only three weeks previously, when she noted that

[1] A paper read before the Medical Society of the State of Pennsylvania, at Williamsport, Pa., May 17, 1893.

her legs were swollen. Shortly after this she became short of breath. She continued, however, to perform the duties of her position up to the time of her admission to the hospital.

On admission her appearance was most striking. A lustrous pallor pervaded her entire body—in fact, she presented the characteristic waxy hue of chronic renal disease. Her feet and legs were much swollen, as were also the right hand and face. To a less degree she was everywhere edematous, though more so on the right side, on which she mostly lay. The urine was dark-hued, had a specific gravity of 1030, was loaded with albumin, and contained numerous granular casts.

My colleagues, as well as myself, all said : a "typical case of renal disease." The urine-examination seemed scarcely necessary. The diagnosis could be made at a glance. As a matter of course, however, her heart was also examined. The apex-beat, in the third interspace, was forcible and rapid, from 108 to 120, while the respirations numbered from 40 to 43 in the minute. On palpation, a cardiac thrill, systolic in time, could, in addition, be appreciated.

Percussion determined the upper border of cardiac dulness to the left of the sternum, at the superior edge of the third costal cartilage ; the right border one inch to the right of the right margin of the sternum. There was, therefore, enlargement of the right side of the heart, as well as of the left. There were the signs of pleuritic effusion on both sides. Auscultation recognized a double murmur, most intense at the apex, with the systolic portion conducted into the axilla.

Here was then a case of mitral disease—regurgitation, and probably stenosis, with right-sided dilatation ; but the intense waxy appearance and the pronounced albuminuria led us to believe that there

was also positive renal disease, probably subacute parenchymatous nephritis.

The girl was ordered absolute rest in bed, five minims of tincture of digitalis three times a day, and caffein citrate, three grains every four hours. Copious diuresis set in, and at the end of five days the dropsy was almost totally gone. The scanty urine had increased until it reached 62 ounces, while the albumin and the casts had entirely disappeared. The change was almost incredible. I had never seen such a striking one in so short a time. The mitral murmur continued, showing that the heart-disease was the permanent one, and the idea, almost irresistible at first, that there was some special form of renal disease, independent of the passive congestion due to the cardiac disease, was dissipated.

She remained in the hospital until December 24th. Once there was a return of the albumin and a few granular casts, concurrent with an attack of bronchitis, but these again disappeared, and she was discharged seemingly quite well. The diastolic part of the double mitral murmur finally also disappeared, and the heart had been restored to its original dimensions, but the mitral systolic murmur continued, and is likely to be permanent.

Case II.—F. R. P., a bank-officer, aged forty-two, was seen in consultation with Dr. S. R. Crothers, of Chester, Pennsylvania, on November 19th, 1892. With the exception of typhoid fever, ten years before, and an attack of influenza in December, 1891, he had never been seriously ill. About one year previously to his visit to me he first began to have attacks of palpitation of the heart and shortness of breath, with headache. He was treated for "functional trouble," and improved sufficiently to return to work. In this, however, he overtaxed himself, and at the end of about two months he was again compelled to give up. Although he spent

much of the summer at the seaside, there was not improvement sufficient to permit his return to business. All of this time his symptoms pointed to heart-disease, and his own belief and that of his family were that this was his principal, if not his sole illness.

At the date of his visit to me there were aggravated dyspnea and cough, but the headache, from which he had before suffered so much, had disappeared. There was also nausea, especially in the morning, but at times also in the day, associated occasionally with diarrhea. His complexion was intensely sallow—almost cachectic.

On inquiry, I found that his feet were a little swollen at night, but that this swelling disappeared each morning. Examination of his heart disclosed enormous hypertrophy of the left ventricle, the apex being lowered and displaced to the left. The aortic second sound was accentuated, but there was *no murmur*. The action of the heart was tumultuous, its rate frequent, and its rhythm irregular.

The urine throughout had been copious, light-hued, and low in specific gravity, until a short time before his visit, when it had become scanty and high-colored; but at the time of his visit, through the action of diluents and diuretics, it was increased to 64 ounces in the twenty-four hours. A portion of this examined by me had a specific gravity of 1012, and contained only one-twentieth per cent. of its bulk of albumin, and a small number of hyaline casts.

In this case the prominent symptoms were cardiac, in contrast with Case I, in which the most striking symptoms pointed to the kidneys as their source, and in fact were at the time caused by the renal complication. Yet the former was primarily

a case of renal disease, and the latter one of heart-disease.

This is a state of affairs that supervenes on an interstitial nephritis, a gradual hypertrophy of the left ventricle, the rationale of which is not perfectly agreed upon, but which I am inclined to look upon as a compensatory hypertrophy, maintaining the individual in tolerably good health until compensation begins to fail, when the symptoms of cardiac distress described set in, and increase until death takes place, as in this case, suddenly, from simple cardiac failure, or, as is perhaps more frequent, from uræmia.

In this instance, therefore, a case of primary renal disease was overshadowed by cardiac symptoms in so marked a degree as to cause the latter to be overlooked.

Case III.—A. G. B., a retired merchant, aged sixty-two, consulted me first on the 16th of April, 1892. He had a clear family history of gout, his father having been gouty years before his death, and dying suddenly. The patient himself had been subject to attacks of gout for a number of years, and had tufaceous deposits in his knuckles. He had an attack of influenza in the early part of 1890, and another in May, 1891; but during the year previous to his consulting me he had been more than usually exempt from gout.

Albuminuria was discovered by Dr. Pepper in May, 1891. Following this the patient spent the summer in Europe. While in Europe the albumin disappeared, and continued absent until a few days before he consulted me, when its presence was discovered by his son, a medical man. He also noted at this time a little dyspnea on exertion, but with

these exceptions felt as well as usual. His last evident attack of gout was on board ship, in the autumn, while returning from Europe.

At the time of his visit to me, in April, 1892, I examined his urine, morning and evening samples. Each contained one-tenth of its bulk of albumin, but I found no casts in either sample. The morning urine had a specific gravity of 1016; the evening, of 1014. I concluded promptly that I had to do with a case of chronic interstitial nephritis of gouty origin.

In June, following his visit to me, the patient again went to Europe, and returned late in September, 1892. He had a couple of slight attacks of gout during his absence, and a little oppression in breathing on shipboard, so that he had to sit up in his berth. During the month of June there was some irregularity of action of his heart, which he ascribed to flatulence. With these exceptions, he considered his health even better while in Europe, in the summer of 1892, than in the previous summer.

On November 21, 1892, he again consulted me. He complained of flatulence, and of what he termed wind pressing on his heart. This prevented him from lying down in comfort. He also thought that it caused cough. On the day before his visit he first noted a little swelling of his feet.

I now examined his urine again. It was dark-hued, contained one-tenth of its bulk of albumin, and this time I found a few hyaline casts. There was also a little swelling about his ankles, and auscultation disclosed an evident mitral systolic murmur. I prescribed rest at home (he had been attending to some business), salicylic acid, pepsin, and aromatic powder for the flatulence; also hot water before meals; and for the dropsy, a pill containing digitalis, quinine, squill, and nux vomica. In a few days the edema had disappeared, and the

dyspnea was partially relieved. A few days later both symptoms returned, and although again relieved for a time, they proved ultimately intractable. There was dulness to the right of the sternum. Evidently the right ventricle was yielding. The urine became more scanty, the albumin increased, as also did the casts, and the patient died suddenly December 20th.

CASE IV.—John W., a colored laborer, fifty-two years old, was admitted to the Hospital of the University of Pennsylvania, April 18, 1893. His family history was negative. He had had a bad attack of typhoid fever thirty-five years ago. Eight years ago, while working in a brick-yard, he suddenly fell, and was unconscious for about four hours. With returning consciousness came unimpaired muscular power; but four years later he awoke one morning completely paralyzed on his left side, without any evidence of anything else happening in the night. At the end of three days, however, he began to regain power in the arm and leg of the affected side, and in three months was able to move around with the aid of a cane. His condition in this respect has been improving steadily, but two years ago he noted that he was short of breath, and last winter this symptom grew worse, while edema of the legs was superadded. These symptoms finally became so aggravated that he entered the Philadelphia Hospital, whence, after six weeks, he was discharged, greatly relieved.

Two weeks previously to admission to the University Hospital he again became ill, the chief symptoms being enormous swelling of the legs, orthopnea, and a dry, hacking cough, very troublesome at night. On admission we noted also irregularity of the heart, hypertrophy of the left ventricle, and an easily recognizable mitral systolic murmur. There was also the remnant of the left-sided paralysis, and

on this side the edema was much greater than on the right side. There were the signs of effusion in the left pleural sac, and the percussion-boundary of the liver indicated some enlargement.

The urine was albuminous, containing one-seventh of its bulk of albumin, but no casts were found.

The diagnosis here made was mitral regurgitation, a diagnosis that was thought to be sustained by the enlargement of the liver. Treatment proved ineffectual, and the man died less than three weeks after admission.

At the necropsy there was found enormous eccentric hypertrophy of the left ventricle, with dilatation of the right ventricle, both ventricles, and especially the right, being distended with blood. The heart weighed 1000 grams, with the blood in it, 750 when emptied, the normal weight being about 337 grams. The *mitral leaflets were a little thickened, but essentially normal.* The aortic cusps were normal, as were also the valves of the right side of the heart.

There was a large effusion in the left pleural cavity, as recognized before death, and a patch of congestion in the lower part of the left lung. The liver weighed 1285 grams, and was *not*, therefore, enlarged, the normal weight being from 1550 to 1860 grams.

Both kidneys were enlarged, the right weighing 210 grams, the left 220 grams, the normal weight being from 130 to 150 grams. The cortices especially were widened, and the seat of chronic parenchymatous nephritis, which, in the light of the autopsy, I regard as the cause of the hypertrophy of the left ventricle.

I have selected, from among many cases, these four of combined cardiac and renal disease, in all of which there was, at the beginning, and in some

throughout life, more or less erroneous conception of the exact state of affairs. The first case, that of the shop girl, aged fourteen, was supposed to be a case of chronic parenchymatous nephritis, while further study showed it to be one of primary valvular disease of the heart, with secondary passive congestion of the kidney.

The second case was one of chronic interstitial nephritis, with hypertrophy of the left ventricle, in which all of the symptoms pointed to primary heart-disease, but the true nature of which was easily revealed upon closer study.

The third case was a more complex one than either of the others. An ancestry of gout; a personal history of much gout; an albuminuria, at first small; absence at first of casts, and then sparse casts, all pointed to gouty kidney, a supposition by no means weakened by the oppression and dyspnea, symptoms that almost always supervene when the hypertrophied left ventricle of chronically contracted kidney begins to fail. On the other hand, the evident mitral murmur, the moderate degree of hypertrophy of the left ventricle, the stretched right ventricle, the irregular heart, the gastric catarrh, and the dropsy, pointed to a condition of mitral regurgitation, which, I cannot but think, was also present, a view in which I was also sustained by Professor Da Costa, who saw the case with me. Yet the problem was a difficult one, and the question, "heart or kidney?" was carefully debated. It seemed to us that in this case doubtless both organs were affected independently of that interdependence that always exists between these two important

organs. That is, there were interstitial nephritis and endocarditis, both the result of the same cause—gout—and the coöperation of these two conditions accounted for the intractableness of the case. Unfortunately, in this case there was no autopsy to settle the question, and it must always remain a matter of probabilities.

That such a state of affairs does occur is shown by the following interesting case:

Case V.—C. K., a Russian coal-miner, aged twenty-eight, from Schuylkill County, was admitted to the Hospital of the University of Pennsylvania, April 12, 1893. On account of his total ignorance of English no family or previous personal history could be elicited, except the indefinite statement that his feet had been swollen for ten years, and yet that he was perfectly well up to three weeks before admission, and working in a coal-breaker.

On admission he was gasping for breath, in an unpleasant, grunting manner. His heart was extremely irregular, and at times beat so rapidly that it could scarcely be counted. It was noted soon after admission at 144, again at 166. His respirations on admission were 36; and when his pulse was 166, the respirations were noted at 46. His temperature was normal. His belly was distended with fluid. There was edema of the feet and legs—in fact, general anasarca—and he lay in such a stupid state that it was difficult to get any information from him. On admission he was expectorating a blood-stained mucus, regardless of time or place.

Physical examination revealed the liver to be much enlarged and tender; the heart not much enlarged, but acting tumultuously, and no murmur could be detected. Subsequent examination showed the right boundary of the right ventricle at the right

edge of the sternum, indicating enlargement of the right heart. The examination of the lungs, on admission, was not satisfactory, because of the extreme illness of the man, and his disgusting, filthy state. The urine was scanty, high-colored, and bile-stained. It contained only a moderate amount of albumin, from one-twentieth to one-eighth of its bulk, but numerous hyaline and pale granular casts.

Under full doses of the infusion of digitalis he improved quite rapidly, the pulse and breathing rate coming down gradually. The rhythm of the pulse also improved, and the anasarca diminished. On the 30th of April his pulse had fallen to 64, and my note reads: "Although the pulse is slow and forcible, no murmur is detectable, yet we feel sure there is mitral regurgitation." His improvement was so great that he was even allowed to sit up. On May 5th, his temperature began to rise, and concurrently his breathing to be more frequent. On the 11th the signs of fluid in the left pleura were apparent (probably had shown themselves a couple of days earlier, because, on account of difficulties, he was not examined daily). The respirations were 50, but the pulse did not rise above 108, and for the first time a distinct mitral systolic murmur was audible. On the 14th it was thought a murmur was heard, but repeated examinations, even when his heart was at its best, failed to detect anything definite until the 11th. The man died suddenly a day later.

The necropsy was most instructive. The lesions were numerous, but most interesting in this connection was the association of extreme mitral stenosis, in the shape of a button-hole mitral orifice, and a pair of typical large white kidneys, beginning to undergo contraction. There was also

enlargement of the heart, more especially in the direction of the right ventricle, as recognized before death, associated with marked dilatation of the left auricle. There was a left-sided empyema of recent origin, as shown by a fresh layer of thick lymph, probably the immediate cause of death; anthracosis, or miner's lung, with cavities; an enlarged hypertrophically cirrhotic liver; an enlarged spleen with two pyoid sacs, probably softened infarcts.

The fourth case was a complete surprise in the light of the autopsy; yet I am doubtful whether, with the situation repeated, I would make a different diagnosis. The distinct mitral systolic murmur and the extreme anasarca, the scanty urine and pleural effusions, all combined to favor the idea that there was mitral regurgitation, while the fact that the man had had two seizures, best explained on the supposition of cerebral embolism, of which a cardiac embolus is the most common cause, seemed to add all that was required to make the diagnosis absolute. One symptom only might have suggested a renal origin, and that was the degree of hypertrophy of the left ventricle, without aortic disease. Mitral regurgitation, while causing hypertrophy of the left ventricle, seldom produces the extreme hypertrophy that attaches to aortic valvular disease or chronic renal disease without aortic valve disease. At the same time the extent of this hypertrophy was not appreciated before death, because of the difficulty of investigation occasioned by the naturally thick chest-walls, further thickened by anasarcous infiltration. Under the circumstances, the mitral murmur is best explained on the supposition that there was

mitral regurgitation consequent upon dilatation of the hypertrophied left ventricle.

Yet, notwithstanding the difficulties encountered in the separation of heart-disease from kidney-disease, there are certain points that, if borne in mind, aid in the discrimination. These points, already referred to, deserve, in conclusion, to be recapitulated: First, the conditions most likely to be associated or confounded are mitral disease on the one hand, and chronic parenchymatous nephritis on the other, or the last stage of interstitial nephritis, when the urine, from having been copious and light-hued, becomes scanty and dark-hued, because of failing cardiac power.

These points of diagnosis refer more particularly to uncomplicated cases. With complicated cases the difficulty is greatly increased, and the decision must be one of probabilities. A fact of great value in my judgment in favor of a primary and advanced kidney-disease, is the failure of heart-tonics like digitalis to produce diuresis, even though the pulse-rate is decidedly reduced by the action of the remedy.

Now, what is the practical bearing of these considerations? I answer, not so much in the direction of therapeutics as in that of prognosis. For, happily, the treatment of these conditions is essentially the same, and to this subject I have given very full consideration quite recently elsewhere.[1] In the

[1] "The Relations between Renal Disease and Disease of the Circulatory System," International Clinics, vol. iv, second series, 1893. "Treatment of Chronic Valvular Disease of the Heart," Therapeutic Gazette, April 15, 1893.

matter of prognosis, however, it is of the extremest importance. For in cases of pure mitral disease, not too far advanced, the most favorable prognosis may be given, as evidenced by our experience with Case I. In the stage of renal disease under consideration, however, and in the combined form with actual structural change in both heart and kidney, the prognosis is very unfavorable, and to mistake the one condition for the other, and to prognosticate accordingly, may seriously jeopardize one's reputation.

Chronic parenchymatous nephritis.	Chronic interstitial nephritis. (Last stages.)	Mitral Disease.
Urine scanty and high-colored; high specific gravity; highly albuminous.	Urine though scanty is still light-hued, and has low specific gravity; moderately or slightly albuminous.	Urine scanty and high-colored; high specific gravity; moderately or slightly albuminous; rarely highly albuminous.
Numerous granular, dark granular or fatty casts.	Few casts, and these hyaline or slightly granular.	Few casts, hyaline or slightly granular.
Much dropsy.	Little dropsy as a rule, though when heart fails dropsy may be marked.	Much dropsy; effusion into serous sacs.
No mitral systolic murmur.	No mitral murmur.	Mitral murmur.
As a rule no hypertrophy of left ventricle, which may, however, be present at times.	Always marked hypertrophy of left ventricle, except in persons feeble and cachectic at the outset; this without aortic murmur.	Moderate hypertrophy of left ventricle; hypertrophy of right ventricle.
No enlargement of liver.	No enlargement of liver.	Enlarged and tender liver.
No signs or symptoms of arterio-capillary fibrosis.	Symptoms and signs of arterio-capillary fibrosis may be present.	No arterio-capillary fibrosis.
No retinitis albuminurica.	Retinitis albuminurica may be present.	No retinitis albuminurica.
No history of gout.	History of gout, lead-poisoning, or free eating and drinking.	Seldom a history of gout, alcoholism, or free eating and drinking.
Seldom a history of rheumatism; more frequent of infectious disease.	No history of rheumatism or infectious disease.	Probable history of rheumatism or infectious disease.
Uremia infrequent.	Uremia frequent.	No uremia.
Partial response to treatment.	Doubtful response to treatment.	Generally prompt response to treatment.